

Periscope

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1. Sexual Excitation in the Anxious Psychopathies. CULLERRE.
2. A New Case of Conjugal Paresis of Syphilitic Origin. CARNIER and SARTENOISE.
3. Epilepsy, Alcoholic Delirium, Melancholia with Suicidal Attempts and General Paralysis in the Son of an Alcoholic Mother and a Father who Suicided, the Patient Himself being a Syphilitic and an Alcoholic. SIMON.

1. *Sexual Excitation in the Anxious Psychopathies.*—A lengthy article dealing with the relationship between the anxiety phobias and sexual excitation. Space forbids an extended consideration. There is at first an association of the two symptoms. Sexual excitation is a common phenomenon in all mental maladies, observable often in the periods of invasion not only organic, but of simple psychoses. It is frequent in the course of the systematized delusions and responsible for many of the delirious ideas, but it plays only an accidental rôle. Predisposition and heredity and emotional disequilibrium have their influence. The author thinks there is a pathogenic bond between the two conditions. The two symptoms are generated by an incidental mechanism. Freud's theory which makes the anxiety a condition consequent upon an accumulated genital tension is not favored by the author. In his cases the anxiety was always the primary phenomenon, then the sexual excitation. Experiment has shown the existence of cortical centers governing the functions and appetites, and in particular the organs of generation, and the author traces the psychologic connection between erotic ideas and sexual excitation, the persistence in consciousness of erotic ideas, but he does not trace the relationship between these and anxiety. There is also a discussion of relative control of the superior cortical centers and the automatic infra-cortical centers, but no explanation is essayed by the author, he desiring merely to show the coincidence of two phenomena which would seem to exclude each other.

2. *A New Case of Conjugal Paresis of Syphilitic Origin.*—Cullerre had previously reported three cases in which, however, the luetic etiology was not unqualifiedly established. In 1904, Cullerre reported a fourth case, in which he thought himself entitled to say: "I consider the last case as favoring the luetic origin of general paralysis." The two writers report a new case further fortifying this position. This case, with those of Cullerre (1890) and cases of conjugal paresis on a luetic basis previously reported by Acker in 1887 and Mendel in 1888, establish in the author's opinion the contention of this article. There had been about forty cases reported up to 1904.

3. *Epilepsy, Alcoholic Delirium, Melancholia with Suicidal Attempts.*—The history of this young man is carefully traced. It shows the unusual association of symptoms of very distinct entities. The influence of the alcoholic and the luetic heredity is discussed. This case must be read in the original. WOLFSTEIN (Cincinnati).

(Vol. 19, 1905, No. III, March.)

1. Secondary Degeneration after Focal Softening in the Calcarine Region. PROF. WEBER.
2. Clinical Study on Stereotypy in Dementia Precox. DROMARD.

1. *Secondary Degeneration after Focal Softening in the Calcarine Region.*—Patient died aged 76 years. Admitted profoundly demented, complete amnesia; no history obtainable. Visual acuity much diminished, the glance is vague. Patient did not fix the persons addressing her. She winked, however, when an object was approached to the eyes, and infrequently she could recognize what was shown her. The dementia precluded any careful analysis. Autopsy.—A focus of softening was found in the right calcarine region, about 40 mm. long. Pal stain was used. The examination was rendered more difficult by reason of an unfortunate cut made during autopsy precisely through the region of the external geniculate bodies. The author agrees with the well-known view of v. Monakow that every destruction of the median part of the occipital lobe must cause a secondary degeneration of the cortical visual neurons. C. Genic Ext. optic rad. cortex. There were found in Weber's case besides the softening in the calcarine region (1) slight areas in corpus callosum, (2) a linear destruction in the hippocampal convolution, (3) one in the convolution gendronnée. All of these were "quantités négligeables." The drawings accompanying the article give a clearer idea of the secondary degeneration. The article deserves careful perusal as a contribution to the study of hemianopsia after focal lesions in the visual area.

The microscopical examination was made with great care, but it is a pity that control specimens with the Marchi method were not made. We give Weber's résumé: Following softening in the calcarine region there was observed: 1. The sagittal fibers were directly injured equally. 2. The secondary degeneration was at first localized in the inferior part of the Optic Radiation and the Fascia Long Inf. 3. This degeneration could be subdivided into three areas: (a) More frontal—an inferior zone tending to the temporal lobe; (b) a superior zone going to C. Genic Ext.; (c) one to Pulvinar. 4.. This degeneration was certainly more extensive in F. L. I. than O. R. Weber also concludes, in agreement with v. Monakow, that the dorsal portion of the sagittal fibers of the occipital lobe are connected with the posterior areas of the thalamus opticus and with the parietal cortex. They have no relations of any importance with the cuneus lobuslingualis or O2. After v. Monakow, therefore, the bundle which unites the visual cortical area with the primary optic centers divides at the height of the C. Genic into three portions: (1) To the area of Corp. Quad. Ant., (2) to the pulvinar, (3) to the C. Genic Exter.; 2 and 3 were degenerated. Weber demonstrated also that the lateral inferior white matter of C. Genic Exter. was intact, and probably not in relation with the optic sphere. As regards the function of F. L. I., Dejerine's view is "that this inferior longitudinal bundle is an association bundle connecting the occipital lobe, and especially the visual sphere, with the temporal lobe." Monakow and Sachs agree with Dejerine. Flechsig (with his usual methods) and Probst (based on study of lesions in man and animal experimentation) view the F. L. I. as a projection bundle connecting the Thal. Optic with the visual cortical zone. Steroklitzki believes that the F. L. I. is partly an association, partly a projection system: (a) The inferior portion connects the occipital to the temporal cortex (associative); (b) the superior goes to the infra-cortical centers. Thal. Op. Pulv. C. Genic Ext., C. Genic Int. and Nucleus Lentic. (projection). With slight modifications, Weber agrees with the latter view. Weber is of the opinion that the localization of the secondary degeneration depended without doubt upon the lesions of the fibers extending from the cortical focus.

2. *Clinical Study on Stereotypy in Dementia Precox.*—This is a very long classification of various types of stereotypy, both of attitudes as well as of various other forms of motorial activity, such as speech, writing, mimicry, the gait and complex acts. There is also a consideration of the evolution of stereotypy. Kraepelin defines this form of activity as the abnormal duration of motor impulses which produce a permanent contracture of certain muscle groups, or a repetition of such a movement. The term contracture as used here in the spirit of the author would seem to indicate that physiological state presided over the symptom. The direct modification of cerebro cellular activity under the influence of some presumed toxin conditioned the morbid process, as shown by the prolonged attitudes or repeated movements of the subject. Others, particularly Cahen, would rather consider these states as not being convulsive in character, but as actions which were at first voluntary and conscious, and which later become automatic and subconscious by this fact of long duration and repetition. Here we are not concerned with an active manifestation, with the phenomenon of irritability belonging to the domain of primitive automatism, but it is a residual manifestation of a mind habit belonging to the domain of secondary automatism. We are, therefore, in the presence of a problem in every way comparable to the distinction between the tics and spasms, a distinction well cleared up by Meige and Feindel.

It would also appear to be necessary to distinguish with as much precision as the nature of so complex a subject permits between active manifestations and those of the subconscious residual period. The writer bases his views on the microscopical researches of Klippel, who describes first the immediate lesions in which there is a granular degeneration of the cells, and so-called consecutive lesions, which are characterized by the destruction of connections between different territories or the different cells of the cortex through atrophy of their prolongations, which bring about intercommunication. The article is not well adapted for review. The author also discusses the value of stereotypy as a diagnostic sign, and is not inclined to look upon it as specially peculiar to dementia precox, being found in other forms as well. The prognostic significance, as well as the relation between convalescence and the disappearance of stereotypy, are also discussed.

WOLFSTEIN (Cincinnati).

Nouvelle Iconographie de la Salpêtrière

(Mars-Avril, 1905, No. 2.)

1. Friedreich's Disease and Hereditary Cerebellar Ataxia. RAYMOND.
2. Alterations in the Cerebral Tissue Due to the Presence of Tumors. WEBER, PAPADAKI.
3. Syndrome of the Peduncular Calotte. GRUNER, BERTOLOTTI.
4. A Case of Acromegaly with Hyperplastic Lesions of the Pituitary Body, Thyroid Body and of the Suprarenal Bodies. BALLET, LAIGNEL-LAVASTINE.
5. Cyanosis of the Retina in Sclerosis of the Pulmonary Artery. BABINSKI, MILE. TUFESCO.
6. Acute and Circumscribed Chronic Edema of Nervous Origin. VALOIRA.
7. A Case of Early Thoracic Deformity Following an Acute Pleurisy. MAITIGNON.
8. Professional Cramp and its Treatment by Massage and Reëducation. KOUINDJY.

1. *Friedreich's Disease.*—Raymond's conclusion from the study of these cases and from the pathological findings in one of them is that it is practically impossible to ascribe all cases of this symptom complex to one morbid entity; that is, to an isolated atrophy of the cerebellum. The